Gastroenterol Hepatol. 2016;39(5):305–310

Gastroenterología y Hepatología

www.elsevier.es/gastroenterologia

ORIGINAL ARTICLE

Vitamin D deficiency in chronic liver disease, clinical-epidemiological analysis and report after vitamin d supplementation☆

Nereida Fernández Fernández*, Pedro Linares Torres, Diana João Matias, Francisco Jorquera Plaza, Jose Luis Olcoz Goñi

Servicio de Aparato Digestivo, Hospital Universitario de León, León, Spain

Received 25 June 2015; accepted 2 October 2015
Available online 30 April 2016

KEYWORDS
Vitamin D;
Cirrhosis;
Chronic liver disease;
Supplementation;
Child–Pugh;
Liver function

Abstract
Introduction: Vitamin D (VD) is known to have multiple extra-skeletal health functions. There is emerging interest in exploring the relationship between vitamin D and chronic liver disease (CLD).
Objectives: To determine the prevalence of VD deficiency in patients with CLD in our setting and to assess whether VD supplementation influences plasma levels and is associated with improved liver function.
Material and methods: We conducted a study in 2 phases. First, we analysed clinical and epidemiological characteristics in 94 patients with CLD; second, different doses of calcifediol (25-OH-VD) were administered to patients with VD deficiency (< 20 ng/mL) and insufficiency (20–30 ng/mL). Plasma concentrations and liver function (Child–Pugh and MELD) at the end of treatment were compared with baseline data.
Results: Deficient or insufficient VD levels were found in 87% of the patients, with an average concentration of 18.8 ng/mL. Levels were lower in patients with cirrhosis (15.9 ng/mL) ($p = 0.002$) and in alcoholic liver disease. VD levels were inversely proportional to the degree of liver function: Child A (16.52 ng/mL) vs Child C (7.75 ng/mL). After VD supplementation, optimal serum levels were achieved in 94% of patients and significant improvements were observed in platelet count, albumin levels ($p < 0.05$) and functional status assessed by the Child–Pugh scale ($p < 0.05$).

☆ Please cite this article as: Fernández Fernández N, Linares Torres P, João Matias D, Jorquera Plaza F, Olcoz Goñi JL. Déficit de vitamina D en la enfermedad hepática crónica, análisis clínico epidemiológico y tras aporte vitamínico. Gastroenterol Hepatol. 2016;39:305–310.

* Corresponding author.
E-mail address: nereidaff@gmail.com (N. Fernández Fernández).

Conclusion: Given the high prevalence of VD deficiency or insufficiency, the need for screening should be considered in the population with CLD. VD supplementation could be safe and effective.

PALABRAS CLAVE
Vitamina D;
Cirrosis;
Enfermedad hepática crónica;
Aporte;
Child-Pugh;
Función hepática

Déficit de vitamina D en la enfermedad hepática crónica, análisis clínico epidemiológico y tras aporte vitamínico

Resumen
Introducción: La vitamina D (VD) participa en multitud de funciones extraesqueléticas en el organismo y cada vez es más importante su relación con las enfermedades hepáticas crónicas (EHC).
Objetivos: Analizar la prevalencia de déficit o insuficiencia de VD en los pacientes con EHC de nuestra área. Evaluar si el aporte de VD influye en la concentración sérica y se asocia a mejoría de la función hepática.
Material y métodos: Realizamos un estudio en 2 fases. En el primer tiempo se analizaron características clínico-epidemiológicas de 94 pacientes con EHC; en un segundo tiempo, se administraron diferentes dosis de calcifediol (25-OH-VD) a aquellos pacientes con déficit (< 20 ng/mL) e insuficiencia (20-30 ng/mL) de VD. Se determinaron concentraciones plasmáticas, variables analíticas y de función hepática (Child-Pugh y MELD) al finalizar el tratamiento y se compararon con los datos basales.
Resultados: El 87% de los pacientes tenían concentraciones deficitarias o insuficientes de VD, con una media de 18,8 ng/mL, siendo menor en los cirróticos (15,9 ng/mL) ($p = 0,002$) y en la etiología por alcohol. Igualmente la concentración sérica de VD era inversamente proporcionales al grado de función hepática: Child A (16,52 ng/mL) vs. C (7,75 ng/mL). Tras el aporte con VD, se consiguió normalizar los niveles en el 94% de los pacientes, mejorar significativamente la cifra de plaquetas, de albúmina ($p < 0,05$) y el grado funcional valorado por la escala de Child-Pugh ($p < 0,05$).
Conclusión: Dada la alta prevalencia de déficit o insuficiencia de VD debería plantearse la necesidad de cribado en la población con EHC. El aporte de VD podría ser seguro y eficaz.

Introduction

Vitamin D (VD) is a lipid-soluble vitamin which, in addition to being an essential micronutrient, can also be considered a hormone involved in a complex system that regulates mineral homeostasis, protects skeletal integrity, and modulates cell growth and differentiation.[1]

We are currently witnessing a worldwide silent epidemic of VD deficiency. There is a widespread, though not undisputed, consensus that normal serum VD concentrations should be between 30–50 ng/mL. VD insufficiency is considered to be levels of between 20 and 30 ng/mL, and deficiency occurs when these fall below 20 ng/mL.[2] More than 1 billion people worldwide are estimated to have VD deficiency or insufficiency, with a higher risk in elderly patients or those with chronic diseases, such as chronic liver disease (CLD) or inflammatory bowel disease (IBD).[3,4] Insufficient VD levels (20–30 ng/mL) are almost universal in patients with CLD, and approximately two-thirds of this population have deficient levels (<20 ng/mL). These levels drop further still in the case of advanced cirrhosis, and as hepatic dysfunction becomes more severe.[5,6]

There is growing interest in the non-classical or extraskeletal functions of VD. Insufficient VD levels in CLD have been associated with an increase in bacterial infections,[7] complications due to portal hypertension, severity of liver fibrosis and mortality.[8] Given the increasingly important link between VD and liver disease, it seems appropriate to explore this relationship in patients with advanced CLD.

Our primary objective was to determine the prevalence of VD insufficiency or deficiency in patients with CLD in our health area, and to examine whether VD differed according to the aetiology of the CLD, presence of liver cirrhosis or grade of liver failure. In a second phase, we also examined whether VD supplementation could correct deficient levels, and if this led to an improvement in the CLD functional class.

Materials and methods

All patients with CLD seen consecutively in the specialised liver clinic of our tertiary hospital (Complejo Asistencial Universitario de León, Spain) during the months of March and April 2014 were included in the study.